saccharine is sufficient to cover the taste of 10 gr. of calcium chloride. This mixture may be given either in water or in milk, and it does not interfere with the use of any other remedies.

I have also used it in cardiac disease, where the ventricular wall appeared to be losing power, and here also the results have been encouraging. In cases of pneumonia, where one wishes to get rapid action, I think the chloride is the best salt of calcium, but in cardiac disease other salts may be employed, such as the lacto-phosphate or the glycero-phosphate. It is quite possible that the great benefit one frequently observes from a milk diet in cases of heart disease, may be due, in part at least, to the large quantity of calcium salts which the milk contains.

At the present moment, when there is a good deal of antivivisection agitation, it may be interesting to note that the plan of treatment by oxygen and strychnine recommended by Dr. Prickett and myself fifteen years ago, and the one which I now recommend, both owe their origin to laboratory experiments, and any benefits which may result to patients from either plan must be regarded as the fruits of experiments upon animals.

DELAYED CHLOROFORM POISONING.

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THE following are notes of 2 cases in which symptoms of delayed chloroform poisoning were observed in adults.

CASE I.

A. B., aged 26, was sent to the Western Infirmary by Dr. Thomson on March 2nd, 1906, suffering from acute appendicitis. She was operated on an hour and a half after her arrival and a large diseased appendix removed. The temperature fell to normal the next day, but persistent vomiting continued for two days in spite of washing out the stomach and rectal feeding. On the fourth day after operation she complained of pain in the cardiac region, which was not relieved by strychnine or digitalin, and she died on the fifth day after operation.

I am indebted to Professor Muir for notes of the *post-mortem* examinations in this and the following case.

Post-mortem Examination.—There was no trace of peritonitis, and the site of operation showed normal healing over the point where the appendix had been removed. The stomach contained a considerable quantity of altered blood, but there was no ulceration, and no source of the haemorrhage could be detected. The liver was in a condition of extreme fatty degeneration, the colour being almost a uniform yellow. The organ was not enlarged. The kidneys showed a diffuse fatty degeneration, though rather less marked than was in the liver. The cardiac muscle also showed considerable fatty change, this being pretty generally distributed. The coronary arteries were healthy. The lungs had a distinct acetone-like odour. The spleen was healthy. Microscopic examination gave confirmative results. In the kidneys the convoluted tubules, Henle's tubules, and also many of the straight tubules showed profound fatty degeneration, the cells being practically filled with small fatty globules. The fatty change in the liver was also very advanced and almost uniform in its distribution. In the heart the fatty degeneration was very diffuse, practically all the fibres containing fatty globules, but was not of extreme degree. The changes noted correspond with those in late chloroform poisoning, and point to the action of some toxic substance on the organs mentioned. There was no evidence of septicaemia or of any other condition which might explain the fatal result.

CASE II.

B. C., aged 28, was sent to the Western Infirmary by Dr. Ingram of Helensburgh. She was suffering from pyloric stenosis, and for this a gastro-enterostomy was performed on November 11th. After the operation she was much troubled with sickness and faintness, and died on November 15th. She had as anaesthetic 5 c.cm. ethyl chloride and 3½ oz. of A.C.E. mixture. The anaesthesia lasted for fifty minutes.

Post-mortem Examination.—A posterior gastro-enterostomy had been performed, and the peritoneum was in a healthy condition. The stomach showed signs of chronic catarrh, and its mucous membrane was somewhat rugose. There was no haemorrhage. The liver showed extreme fatty degeneration as in the previous case, and microscopic examination showed the condition to be of similar nature and degree. In the kidneys fatty change was present, but it was much less marked than in the previous case; and it was found on microscopic examination that the fatty globules were only present in the basal portions of the cells. The spleen was normal, and cultures made from it gave only a few colonies of the *Bacillus coli*. The heart muscle was somewhat atrophied, but was not fatty. In the right lung there was an area of chronic grey tubules with evidence of caseation at places, but this was of small extent.

In the second case the changes in the liver and kidneys were somewhat similar to those in the first, but were of less degree. The case is possibly of the same nature, but considerable emaciation was present, and death may have resulted simply from inanition.

I record these cases owing to the importance of cases of delayed chloroform poisoning and the interest that has recently been taken, more especially regarding deaths among children. In the first case the patient had chloroform alone, in the second case ethyl chloride and A.C.E. mixture was given. This latter combination may have accounted for the symptoms in the second case not being quite so pronounced. So many feeble patients have operations done of even a more serious nature than the above and get perfectly well, that it is to a certain extent satisfactory to be able to give a definite cause as to the fatal termination in two such comparatively young patients as the above. The practical question naturally arises, What is to be done in order to prevent the possibility of acetone poisoning taking place?

CHRONIC PERITONITIS CAUSING ELEPHANTIASIS.

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A WOMAN of 43 years of age was admitted to Westminster Hospital under the care of Dr. de Havilland Hall, to whom we are much indebted for permission to publish the case, on September 15th, 1905. She complained of shortness of breath and swelling of the legs and abdomen.

She had been an in-patient at several other London hospitals, and we are much indebted to Dr. Jex-Blake for kindly allowing us to make abstracts of his notes taken during the patient's stay in St. George's Hospital.

HISTORY.

Patient has had five miscarriages and ten children; the menses ceased fifteen months ago. She denies alcoholic excess. In March, 1904, she began to suffer from dyspnoea on exertion, loss of strength and flesh, and languor; in April from vomiting; in June and July from protrusion of the eyeballs; in July, swelling of the thyroid gland; about the same time she complained of nervousness, and the colour of the skin became darker; in August the abdomen became enlarged; in October she suffered from bronchitis and palpitation. A diagnosis of Graves's disease was made. In January, 1905, the following was noted: Abdomen enlarged, walls thick and brawny; no evidences of ascites; lower extremities large, brown stained, and in a condition of hard oedema; no albumen in urine. In February patient suffered from diarrhoea, which continued for two months. On April 9th condition was as follows: Orthopnoea; pulse irregular, small, of low tension; apex beat fourth and fifth spaces, 4 in. to 4½ in. from middle line; mitral systolic bruit conducted toward axilla; jugular veins distended and fill from below; signs of fluid at both bases, and scattered rhonchi; tongue small, red, and raw; abdominal wall hard and irregularly pits; legs much swollen, very tense; skin of body regularly pigmented; moderate exophthalmos; moderate, non-pulsating, bilateral goître. In June there was oedema up to the level of axilla, evidences of ascites, the legs were of brick-red colour, brawny, and barely pitted.

On admission to Westminster Hospital the patient presented the following condition: Orthopnoea; moderate exophthalmos; movements of eyeballs very quick; thyroid gland moderately enlarged, soft, and elastic, with slight pulsation and thrill; severe palpitation on excitement; pulse 100 per minute, irregular, rather full, low tension; apex beat in fifth interspace, diffuse and feeble; loud, blowing, mitral systolic bruit propagated to axilla; fainter bruit in pulmonary area; carotids pulsating vigorously; jugular veins distended and refill from below; much thirst, tongue dry; abdomen extremely distended, walls in lower part hard and brawny; signs of free fluid in cavity; liver not palpable, upper border at level of fourth rib; external haemorrhoids; lower limbs enormously enlarged and condition well represented by photograph; calf measures 20 in.; skin obviously much thickened, does not pit, hard and brawny, sulci deep, general surface shows closely aggregated minute prominences, colour bronzed, with reddish tint; skin of body bronzed; no pigmentation of mucous membranes; scattered rhonchi over lungs; patient highly nervous and flushes; fine tremors of upper limbs; urine normal.